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1. Pflügers Arch 1996 Feb;431(4):571-7

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## Natriuretic peptides increase a K<sup>+</sup> conductance in rat mesangial cells.

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Mesangial cells (MC) are a main target of natriuretic peptides in the kidney and are thought to play a role in regulating glomerular filtration rate. We examined the influence of cGMP-generating (i.e. guanosine 3',5'-cyclicmonophosphate) peptides on membrane voltages ( $V_m$ ) of rat MC by using the fast whole-cell patch-clamp technique. The cGMP-generating peptides were tested at maximal concentrations ranging from 140 to 300 nmol/l. Whereas human CNP (C natriuretic peptide), rat guanylin and human uroguanylin had no significant effect on  $V_m$  in these cells, human BNP (brain natriuretic peptide), rat CDD/APN-99-126 (cardiodilatin/atrial natriuretic peptide) and rat CDD/APN-95-126 (urodalatin) hyperpolarized  $V_m$  significantly by  $1.6 \pm 0.4$  mV (BNP,  $n=8$ ),  $3.7 \pm 0.3$  mV (CDD/APN-99-126,  $n=25$ ) and  $2.8 \pm 0.4$  mV (urodilatin,  $n=9$ ), respectively. The half-maximally effective concentration (EC50) for the latter two was around 400 pmol/l each. This hyperpolarization could be mimicked with 0.5 mmol/l 8-bromo-guanosine 3',5'-cyclic monophosphate (8-Br-cGMP) and was blocked by 5 mmol/l Ba<sup>2+</sup>. The K<sup>+</sup> channel blocker 293 B (100 micromol/l) depolarized basal  $V_m$  by  $4.3 \pm 0.4$  mV ( $n=8$ ), but failed to inhibit the hyperpolarization induced by CDD/APN-99-126 (160 nmol/l) ( $n=8$ ). The K<sup>+</sup> channel opener cromakalim (10 micromol/l) neither influenced basal  $V_m$  nor altered the hyperpolarization induced by 160 nmol/l CDD/APN-99-126 ( $n=8$ ). Adenosine (100 micromol/l) hyperpolarized  $V_m$  by  $13.4 \pm 1.3$  mV ( $n=16$ ). At 100 micromol/l, 293 B did not inhibit the adenosine-induced hyperpolarization ( $n=6$ ). At 160 nmol/l, CDD/APN-99-126 enhanced the adenosine-induced hyperpolarization significantly by  $1.5 \pm 0.6$  mV ( $n=10$ ). CDD/APN-99-126 (160 nmol/l) failed to modulate the value to which  $V_m$  depolarized in the presence of 1 nmol/l angiotensin II ( $n=10$ ), but accelerated the repolarization to basal  $V_m$  by  $49 \pm 20\%$  ( $n=8$ ). These results indicate that the natriuretic peptides CDD/APN-99-126, CDD/APN-95-126 and BNP

hyperpolarize rat MC probably due to an increase of a K<sup>+</sup> conductance. This effect modulates the voltage response induced by angiotensin II. The natriuretic peptide-activated conductance can be blocked by Ba<sup>2+</sup>, but not by 293 B and cannot be activated by cromakalim. This increase in the K<sup>+</sup> conductance seems to be additive to that inducible by adenosine, indicating that different K<sup>+</sup> channels are activated by these hormones.

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